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Original Article

The relation of anthropometric measurements and insulin resistance in patients with polycystic kidney disease

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ABSTRACT

Objective: To examine the frequency of insulin resistance (IR) and its relation with anthropometric measurements in patients with autosomal dominant polycystic kidney disease (ADPKD). **Material and Methods:** Nonobese 82 patients with ADPKD and 58 age matched healthy controls were enrolled into the study. None of participants were diabetic or receiving renal replacement therapies (RRT). IR was determined by homeostasis model assessment of insulin resistance (HOMA-IR) formula. Tanita body composition analyzer was used for anthropometric measurements. Creatinine clearance of participant were assessed by the modification of diet in renal diseases (MDRD). **Results:** Patients with ADPKD had significantly higher level of urea and creatinine, microalbuminuria, and lower level of MDRD. Body fat distribution and HOMA-IR in both the groups were similar. Systolic and diastolic blood pressure of patients were higher than those of controls. **Conclusion:** We failed to determine a higher frequency of IR among patients with ADPKD.

Key words: polycystic kidney disease, insulin resistance, anthropometric measurements, obesity, diabetes mellitus

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INTRODUCTİON

Autosomal dominant polycystic kidney disease (ADPKD) is the most common hereditary kidney disease which is associated with progressive deterioration of renal functions.[1] End-stage renal disease (ESRD) is observed in 25% of patients aged <50 years and 50% of patients aged <60 years.[2] Insulin resistance (IR) accompanies diabetes mellitus (DM), hypertension, dyslipidemia, glucose intolerance, and hyperuricemia in metabolic and cardiovascular disorders (CVDs).[3] There are controversial reports in terms of IR in patients with ADPKD.[4,5]

Obesity is a risk factor for IR; however, quantity and distribution of body fat are important determinants of obesity-related risk factors.[6] Abdominal or visceral fat is associated with higher incidence of IR and cardiovascular (CV) problems.[7] Anthropometry is a term used to define the measurements of specific body segments or regions. Anthropometric measurements including height, weight, body mass index (BMI), and body fat distribution provide valuable data regarding the future risk of diabetes and CVDs.[8]

We aimed to examine the relation of IR and anthropometric measurements in patients with ADPKD.

PATIENTS AND METHODS

Patients

Eighty two patients with ADPKD admitted to Nephrology outpatients service of Bagcilar Education and Research Hospital between February and April 2015 were enrolled. Fifty eight age, gender, and BMI matched healthy individuals, including 25 males and 33 females, were assigned to control group. Exclusion criteria were DM, pregnancy, malignancy, additional chronic disorder, or patients receiving renal replacement therapies (RRT). None of the participants were under special dietary restrictions. The participants underwent physical examination, and demographic features of the participants were recorded. All the subjects read and signed the informed consent forms before enrolling into the study. The ethics committee of the Bagcilar Education and Research Hospital approved the study.

Methods

Blood samples were collected after a 12-h fasting period. Biochemical variables including serum glucose, urea, creatinine, uric acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), calcium, sodium, potassium, total protein, albumin, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglyceride were analyzed by photometric method on the Siemens Advia 1800 device (Siemens Healthcare Diagnostics, Kobe, Japan). Hormone parameters including HbA1c, insulin, and thyroid-stimulating hormone (TSH) were analyzed by chemiluminescence immunoassay method on a Siemens Advia Centaur device (XE-5000, Sysmex Corp., Kobe, Japan). Urine analysis was performed by spectrophotometric method on a Siemens Advia 1800 device (XE-5000, Sysmex Corp., Kobe, Japan). Hemogram was performed in blood samples that were taken into tubes with ethylenediaminetetraacetic acid using an automatic blood counter (XE-5000; Sysmex Corp, Kobe, Japan). Biochemical and hormonal parameters were examined simultaneously in this study.

Insulin resistance was determined by homeostasis model assessment of insulin resistance (HOMA-IR) formula as defined by Matthews *et al*.[9] The normal value for HOMA-IR was considered to be 2.1–2.5. HOMA-IR value of >2.5 was considered glucose intolerance and DM. The following formula was used to determine HOMA-IR: HOMA-IR= [Fasting plasma insulin (U/mL) × Fasting blood glucose (mg/dL)]/405; HOMA-IR= [Fasting plasma insulin (U/mL) × Fasting blood glucose (mmol/L)]/22.5.

Glomerular filtration rate was measured by the modification of diet in renal disease (MDRD) formula (Glomerular filtration rate: 186 × Serum creatinine –1.154 × age – 0.203 × genderrace).

Height and weight of the participants were measured using Tanita Body Composition Analyzer (Tanita Corporation of America, IL, USA). BMI was calculated as weight/height^2 (kg/m^2).

Urine albumin excretion rate exceeding 20 µg/min (30 mg/day), in the absence of uncontrolled hypertension or urinary tract infection, was defined as microalbuminuria.

Statistical analysis

SPSS 22.0 (SPSS, Chicago, Illinois, USA) package program was used for statistical analysis. Parametric variables were compared with independent *t*-test, ordinal data were compared with Mann Whitney U test, and non-parametric variables were compared with chi-square test. Quantitative parameters were compared with Kruskal–Wallis test. Spearman's rho correlation test was used to evaluate the relation of parametric variables. A *P* value of <0.05 was considered significant.

RESULTS

A total of 82 patients with ADPKD, 34 male and 48 female, and 58 healthy subjects, 25 male and 33 female, as controls were enrolled into the study. Demographic properties of study group were shown in Table 1.

Patients group had significantly higher level of urea, creatinine, and uric acid when compared to controls ($P = 0.001$, $P = 0.001$, and $P = 0.002$; respectively). Patients had significantly lower MDRD and urine density ($P = 0.001$ and $P = 0.022$, respectively).

Table 1: Demographic features of study group

	Patients	Controls	^aP
	Ort ± SS	Ort ± SS	
Age (years)	47.71 ± 15.32	46.69 ± 6.74	0.595
Height (cm)	163.73 ± 9.39	163.9 ± 10.48	0.924
Weight (cm)	74.77 ± 13.37	71.65 ± 8.76	0.103
BMI (kg/m^2)	27.95 ± 5.19	26.71 ± 2.03	0.056
	n (%)	*n* (%)	^bP
Gender			
Male	34 (41.5)	25 (43.1)	0.847
Female	48 (58.5)	33 (56.9)	

[a]Student's *t*-test; [b]Chi-square Test. BMI, body mass index.

On the other hand, proteinuria and microalbuminuria-to-creatinine ratio of patients were significantly higher ($P = 0.004$ and $P = 0.001$, respectively) (Table 2).

Body fat and liquid distribution of groups were similar ($P > 0.05$)(Table 3).

The mean systolic and diastolic blood pressure of male patients were significantly higher than that of controls ($P = 0.013$, $P < 0.05$; $P = 0.007$, $P < 0.01$). Male controls had significantly higher MDRD when compared to male patients ($P = 0.001$, $P < 0.01$). Male patients had significantly higher level of urea, creatinine, and uric acid and higher microalbuminuria-to-creatinine ratio ($P = 0.018$, $P = 0.001$, $P = 0.029$, and $P = 0.005$, respectively). Urine density and mean body impedance of male patients were lower than those of male controls ($P = 0.005$ and $P = 0.046$, respectively) (Table 4)

Table 2: Clinical characteristics of patients and controls

	Patients	Controls	*P*
	Mean ± SD (Median)	Mean ± SD (Median)	
Systolic blood pressure (mmHg)	123.66 ± 12.93 (120.00)	121.79 ± 12.72 (120.00)	[a]0.688
Diastolic blood pressure (mmHg)	76.32 ± 80.00 (80.00)	75.17 ± 9.50 (75.00)	[a]0.672
MDRD	91.26 ± 45.26	121.09 ± 27.95	[b]0.001**
HOMA-IR (μU/mmol)	2.89 ± 1.88 (2.58)	2.59 ± 1.84 (1.97)	[a]0.209
HbA1c (%)	5.53 ± 0.39	5.49 ± 0.40	[b]0.627
Glucose (mg/dL)	95.93 ± 10.41	93.93 ± 9.43	[b]0.246
Urea (mg/dL)	42.79 ± 25.05 (33.55)	27.49 ± 8.19 (26.20)	[a]0.001**
Creatinine (mg/dL)	1.23 ± 0.95 (0.93)	0.73 ± 0.15 (0.72)	[a]0.001**
Uric acid (mg/dL)	5.23 ± 1.73	4.49 ± 1.05	[b]0.002**
AST (U/L)	18.26 ± 7.48 (17.00)	19.11 ± 6.16 (17.00)	[a]0.172
ALT (U/L)	16.25 ± 7.82 (14.00)	19.27 ± 10.76 (17.00)	[a]0.071
LDH (U/L)	190.90 ± 72.63 (186.00)	194.95 ± 29.07 (187.00)	[a]0.078
TG (mg/dL)	139.38 ± 74.18 (128.00)	140.45 ± 96.41 (108.00)	[a]0.476
Total cholesterol (mg/dL)	201.31 ± 48.76	194.98 ± 44.33	[b]0.436
HDL-c (mg/dL)	53.19 ± 12.98	52.93 ± 16.31	[b]0.916
LDL-c (mg/dL)	126.34 ± 42.61	116.20 ± 37.13	[b]0.148
Ca (mg/dL)	9.50 ± 0.57	9.43 ± 0.51	[b]0.449
Na (mEq/L)	140.23 ± 15.60 (142)	141.14 ± 2.07 (141)	[a]0.055
K (mEq/L)	4.51 ± 0.44	4.42 ± 0.41	[b]0.177
Total protein (g/dL)	7.33 ± 0.51	7.27 ± 0.39	[b]0.482
Albumin (g/dL)	4.54 ± 0.37	4.58 ± 0.28	[b]0.527
TSH (mU/L)	2.09 ± 1.26 (1.75)	2.72 ± 2.75 (2)	[a]0.367
Insulin (mU/L)	12.03 ± 7.53 (11.03)	11.08 ± 7.93 (8.79)	[a]0.232
WBC (/mm^3)	7.12 ± 1.85 (7.06)	7.25 ± 2.20 (6.97)	[a]0.929
HGB (g/dL)	13.49 ± 1.77	13.89 ± 1.66	[b]0.172
HCT (%)	41.46 ± 5.11	42.64 ± 4.57	[b]0.164
PLT (/mm^3)	241.29 ± 62.38	249.86 ±59.89	[b]0.417
MPV (fL)	7.87 ± 1.31	8.16 ± 1.80	[b]0.267
Urine density (kg/cm^3)	1013.24 ± 7.19	1015.93 ± 6.11	[b]0.022*
Urine protein (mg/dL)	0.29 ± 0.66 (0)	0.02 ± 0.14 (0)	[a]0.004**
Spot creatinine (mg/dL)	131.19 ± 76.35	150.13 ± 72.03	[b]0.141
Urine MA/KR (mg/g)	90.9 ± 174.6 (17.5)	6.80 ± 7.56 (3.95)	[a]0.001**
Urine microalbumin (mg/day)	8.41 ± 13.53 (2.42)	1.10 ± 1.73 (0.52)	[a]0.001**
Fat (kg)	20.97 ± 10.65	19.46 ± 6.02	[b]0.229
Fat (%)	27.16 ± 11.22	27.42 ± 8.43	[b]0.879
Body impedance (ohm)	546.21 ± 79,93	573.72 ± 59.67	[b]0.030*
Body muscle (kg)	28.35 ± 5.35	27.81 ± 4.79	[b]0.542
Body fat (kg)	10.41 ± 5.52	9.96 ± 3.27	[b]0.556
Metabolic age (years)	49.19 ± 16.7	45.40 ± 8.15	[b]0.084
Liquid (kg)	39,28 ± 7.09	38.09 ± 7.21	[b]0.339
Liquid (%)	53.29 ± 8.23	53.09 ± 6.15	[b]0.876

[a]Mann–Whitney U test; [b]Student's *t*-test; *$P < 0.05$; **$P < 0.01$. MDRD, modification of diet in renal diseases; HOMA-IR, homeostasis model assessment of insulin resistance; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; TG, triglycerides; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; TSH, thyroid-stimulating hormone; WBC, white blood cell count; HGB, hemoglobin; HCT, hematocrit; PLT, platelet; MPV, mean platelet volume.

Table 3: Fat and liquid distribution in patients and controls

		Patients n (%)	Controls n (%)	*P*
Fat	<20%	22 (28.2)	12 (21.1)	0.559
	20–27%	16 (20.5)	15 (26.3)	
	>27%	40 (51.3)	30 (52.6)	
Liquid	<50%	27 (34.6)	23 (40.4)	0.738
	50–60%	34 (43.6)	24 (42.1)	
	>60%	17 (21.8)	10 (17.5)	

Chi-square test; Body fat ratio: <20% low, 20-27% normal, >27% high; Body liquid ratio: <50% low, 50-60% normal, >60% high.

Table 4: Clinical characteristics of male patients and control subjects

Male participants	Patients	Controls	*P*
	Mean ± SD (Median)	Mean ± SD (Median)	
Systolic blood pressure (mmHg)	125.59 ± 10.85 (125.00)	117.00 ± 11.99 (120.00)	[a]0.013*
Diastolic blood pressure (mmHg)	76.41 ± 5.52 (80.00)	71.20 ± 8.81 (70.00)	[a]0.007**
MDRD	72.19 ± 29.67	100.51 ± 16.31	[b]0.001**
HOMA-IR (µU/mmol)	2.95 ± 1.97 (2.48)	2.77 ± 1.93 (2.38)	[a]0.742
HbA1c (%)	5.49 ± 0.35	5.52 ± 0.39	[b]0.734
Glucose (mg/dL)	95.65 ± 11.25	93.88 ± 8.29	[b]0.509
Urea (mg/dL)	45.21 ± 26.71 (33.75)	30.8 ± 9.02 (29.00)	[a]0.018*
Creatinine (mg/dL)	1.44 ± 1.11 (1.08)	0.85 ± 0.13 (0.83)	[a]0.001**
Uric acid (mg/dL)	5.64 ± 1.16	5.01 ± 0.96	[b]0.029*
AST (U/L)	18.38 ± 8.39 (17.00)	19.74 ± 5.28 (18.00)	[a]0.131
ALT (U/L)	17.21 ± 7.12 (15.50)	22.45 ± 12.99 (19.30)	[a]0.093
LDH (U/L)	201.38 ± 107.66 (180.00)	199.92 ± 29.13 (188.00)	[a]0.076
TG (mg/dL)	146.91 ± 84.68 (127.50)	163.52 ± 98.42 (129.00)	[a]0.570
Total cholesterol (mg/dL)	185.64 ± 38.75	191.76 ± 46.72	[b]0.585
HDL-c (mg/dL)	49.39 ± 11.98	45.01 ± 11.24	[b]0.160
LDL-c (mg/dL)	116.05 ± 32.79	117.44 ± 38.75	[b]0.882
Ca (mg/dL)	9.45 ± 0.58	9.60 ± 0.47	[b]0.282
Na (mEq/L)	143.21 ± 3.47 (143)	142.20 ± 2.18 (142)	[a]0.193
K (mEq/L)	4.61 ± 0.45	4.42 ± 0.40	[b]0.104
Total protein (g/dL)	7.33 ± 0.60	7.41 ± 0.40	[b]0.567
Albumin (g/dL)	4.64 ± 0.41	4.70 ± 0.21	[b]0.455
TSH (mU/L)	2.21 ± 1.57 (1.84)	1.94 ± 1.03 (1.76)	[a]1.000
Insulin (mU/L)	12.28 ± 7.81 (11.20)	11.71 ± 7.74 (9.79)	[a]0.736
WBC (/mm^3)	7.37 ± 1.97 (7.18)	7.58 ± 2.26 (6.95)	[a]0.927
HGB (g/dL)	14.67 ± 1.76	15.31 ± 1.06	[b]0.110
HCT (%)	44.87 ± 4.88	46.53 ± 2.67	[b]0.129
PLT (/mm^3)	228.66 ± 54.41	238.04 ± 58.72	[b]0.529
MPV (fL)	7.49 ± 1.14	7.86 ± 1.64	[b]0.309
Urine density (kg/cm^3)	1013.15 ± 6.29	1017.68 ± 5.34	[b]0.005**
Urine protein (mg/dL)	0.35 ± 0.75 (0)	0.05 ± 0.22 (0)	[a]0.077
Spot creatinine (mg/dL)	143.02 ± 78.22	174.82 ± 71.83	[b]0.116
Urine MA/KR (mg/g)	91.65 ± 171.26 (17.50)	8.14 ± 9.46 (4.90)	[a]0.001**
Urine microalbumin (mg/day)	8.06 ± 11.8 (2.72)	1.51 ± 2.42 (0.68)	[a]0.002**
Obesity degree (%)	16.19 ± 11.24 (14.83)	11.85 ± 5.35 (10.85)	[a]0.181
Fat (kg)	16.16 ± 9.22	15.21 ± 4.70	[b]0.611
Fat (%)	19.62 ± 9.13	19.53 ± 5.45	[b]0.959
Body impedance (ohm)	507.18 ± 62.91	538.17 ± 47.34	[b]0.046*
Body muscle (kg)	32.01 ± 5.32	32.46 ± 3.51	[b]0.721
Body fat (kg)	9.16 ± 5.17	9.35 ± 3.33	[b]0.860
Metabolic age (years)	47.29 ± 17.10	42.33 ± 9.24	[b]0.161
Liquid (kg)	45.28 ± 5.80	45.39 ± 4.84	[b]0.939
Liquid (%)	58.81 ± 6.69	58.86 ± 3.98	[b]0.975

[a]Mann–Whitney U test; [b]Student's *t*-test; *$P < 0.05$; **$P < 0.01$. MDRD, modification of diet in renal diseases; HOMA-IR, homeostasis model assessment of insulin resistance; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; TG, triglycerides; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; TSH, thyroid-stimulating hormone; WBC, white blood cell count; HGB, hemoglobin; HCT, hematocrit; PLT, platelet; MPV, mean platelet volume.

The mean MDRD, urea, creatinine, and uric acid of female control subjects were significantly higher than those of patients group (P = 0.001 for all). Female patients had significantly higher proteinuria and microalbuminuria-to-creatinine ratio (P = 0.022 and P = 0.001, respectively). Similarly, female patients had higher mean obesity degree and body liquid mass (P = 0.017 and P = 0.008, respectively). There was no significant difference between female patients and controls in terms of systolic blood pressure, diastolic blood pressure, HOMA-IR, AST, ALT, LDH, triglyceride, Na, TSH, insulin, and leukocyte count (P > 0.05). We failed to demonstrate a significant difference between female patients and controls with regard to HbA1C, glucose, total cholesterol, HDL, LDL, Ca, K, total protein, albumin, hemoglobin, hematocrit, platelet, mean platelet volume, urine density, spot creatinine, fat mass, fat ratio, body muscle mass, body fat mass, metabolic age, and liquid ratio (P > 0.05) (Table 5).

Table 5: Clinical characteristics of female patients and control subjects

Female participants	Patients	Controls	P
	Mean ± SD (Median)	Mean ± SD (Median)	
Systolic blood pressure (mmHg)	122.29 ± 14.18 (120.00)	125.42 ± 12.21 (125.00)	[a]0.118
Diastolic blood pressure (mmHg)	79.94 ± 12.29 (72.50)	78.18 ± 9.14 (80.00)	[a]0.152
MDRD	104.77 ± 49.62	136.68 ± 24.71	[b]0.001**
HOMA-IR (μU/mmol)	2.50 ± 1.91 (2.61)	2.45 ± 1.79 (1.93)	[a]0.144
HbA1c (%)	5.55 ± 0.42	5.47 ± 0.42	[b]0.397
Glucose (mg/dL)	96.13 ± 9.88	93.97 ± 10.33	[b]0.345
Urea (mg/dL)	24.00 ± 2.85 (33.55)	24.98 ± 6.58 (23.70)	[a]0.001**
Creatinine (mg/dL)	0.92 ± 0.37 (0.79)	0.64 ± 0.09 (0.64)	[a]0.001**
Uric acid (mg/dL)	4.94 ± 2.01	4.10 ± 0.94	[b]0.014*
AST (U/L)	19.95 ± 6.09 (16.90)	18.63 ± 6.79 (16.00)	[a]0.773
ALT (U/L)	17.65 ± 8.18 (13.50)	16.86 ± 8.10 (14.00)	[a]0.379
LDH (U/L)	183.48 ± 28.59 (186.00)	191.18 ± 28.89 (186.00)	[a]0.456
TG (mg/dL)	134.04 ± 66.17 (134.50)	122.44 ± 92.35 (93.55)	[a]0.132
Total cholesterol (mg/dL)	212.41 ± 52.35	197.50 ± 42.96	[b]0.185
HDL-c (mg/dL)	55.89 ± 13.1	59.12 ± 17.11	[b]0.343
LDL-c (mg/dL)	133.62 ± 47.36	115.23 ± 36.41	[b]0.067
Ca (mg/dL)	9.54 ± 0.56	9.30 ± 0.51	[b]0.054
Na (mEq/L)	138.12 ± 20.00 (141.00)	140.33 ± 1.59 (140.00)	[a]0.158
K (mEq/L)	4.45 ± 0.43	4.41 ± 0.42	[b]0.698
Total protein (g/dL)	7.33 ± 0.45	7.17 ± 0.34	[b]0.088
Albumin (g/dL)	4.47 ± 0.33	4.48 ± 0.29	[b]0.858
TSH (mU/L)	2.00 ± 0.99 (1.72)	3.30 ± 3.43 (2.10)	[a]0.147
Insulin (mU/L)	11.85 ± 7.40 (10.81)	10.60 ± 8.15 (8.46)	[a]0.199
WBC (/mm^3)	6.95 ± 1.75 (6.79)	7.00 ± 2.14 (6.98)	[a]1.000
HGB (g/dL)	12.65 ± 1.22	12.82 ± 1.14	[b]0.535
HCT (%)	39.05 ± 3.74	39.68 ± 3.31	[b]0.433
PLT (/mm^3)	250.24 ± 66.55	258.82 ± 60.10	[b]0.555
MPV (fL)	8.14 ± 1.36	8.39 ± 1.91	[b]0.490
Urine density (kg/cm^3)	1013.31 ± 7.83	1014.6 1 ±6.40	[b]0.435
Urine protein (mg/dL)	0.24 ± 0.58 (0)	0.10 ± 0 (0)	[a]0.022**
Spot creatinine (mg/dL)	122.81 ± 74.69	131.43 ± 67.34	[b]0.597
Urine MA/KR (mg/g)	90.37 ± 178.73 (18.05)	5.78 ± 5.67 (3.90)	[a]0.001**
Urine microalbumin (mg/day)	8.65 ± 14.75 (1.76)	0.78 ± 0.84 (0.45)	[a]0.001**
Obesity degree (%)	24.25 ± 16.95 (19.21)	14.22 ± 7.68 (15.85)	[a]0.017*
Fat (kg)	24.69 ± 10.26	22.56 ± 4.90	[b]0.232
Fat (%)	32.98 ± 9.04	33.16 ± 4.72	[b]0.912
Body impedance (ohm)	576.36 ± 79.17	599.58 ± 54.62	[b]0.153
Body muscle (kg)	25.52 ± 3.28	24.42 ± 1.80	[b]0.086
Body fat (kg)	11.37 ± 5.65	10.40 ± 3.20	[b]0.341
Metabolic age (years)	50.66 ± 16.42	47.64 ± 6.52	[b]0.272
Liquid (kg)	34.65 ± 3.73	32.78 ± 2.26	[b]0.008**
Liquid (%)	49.01 ± 6.65	48.90 ± 3.44	[b]0.921

[a]Mann–Whitney U test; [b]Student's *t*-test; *P < 0.05;**P < 0.01. MDRD, modification of diet in renal diseases; HOMA-IR, homeostasis model assessment of insulin resistance; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; TG, triglycerides; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; TSH, thyroid-stimulating hormone; WBC, white blood cell count; HGB, hemoglobin; HCT, hematocrit; PLT, platelet; MPV, mean platelet volume.

No significant difference was observed between male patients and controls and female patients and controls in terms of body fat and liquid distribution ($P > 0.05$) (Table 6).

DİSCUSSİON

The present study indicated that patients with PKD have similar resistance to insulin action when compared to healthy population probably depending on the similar body fat distribution. Patients with PKD have higher level of blood pressure and uric acid and microalbuminuria independent from body fat distribution.

There is an increasing trend of high BMI among patients with ADPKD from 1985 to 2002.[10] According to a Polish study, the incidence of metabolic syndrome among patients with ADPKD was 14%, while it was 33% for DM in the study from United States.[10,11] CVD is still the most common cause of death in ADPKD[12]. ADPKD is a systemic disease involving multiple organs and affecting metabolic parameters which make patients with ADPKD candidates of metabolic disorders and CVDs.

Memili *et al.* pointed out the relation between ADPKD and insulin resistance independent from preserved renal functions.[13] Patients with ADPKD with well renal reserve have impaired insulin tolerance test. Turkmen *et al.* showed that IR accompanies ADPKD at the earlier stages of disease[14]. Nowacka *et al.* found an increased rate of IR among patients with ADPKD, especially carriers of some gene mutations.[11] Reed *et al.* revealed out a significant association of hyperinsulinemia, decreased insulin sensitivity, and type 2 diabetes with renal cyst volumes.[10] Also diabetic patients with ADPKD die at a younger age when compared to nondiabetic counterparts with better metabolic control.[10] Contradictorily, Vandana *et al.* failed to determine the association between IR and ADPKD[15]. Similarly, we did not examine a relationship.

Increased adipose tissue is defined as lipid ratio of >19% for men and 22% for women.[16] Several methods were used to assess the obesity degree and the distribution of fat and muscle mass. Anthropometric measurements are useful and noninvasive tools to examine the nutritional status as well as the body fat distribution. These measurement include weight, height, BMI, waist-hip circumference and ratio, calf circumference, overarm length, skinfold thickness, and bioelectrical impedance.

A well-known correlation has been established between body weight and blood pressure. A study by Zhang *et al.* showed that blood pressure control is poorly achieved in patients with BMI of >30 kg/m^2 when compared to nonobese individuals.[17] A weight gain of 5–10 and >25 kg have 1.7- and 5.2-fold increased risk of hypertension, respectively.[18] On the other hand, body fat distribution, especially abdominal obesity, is associated with higher risk of hypertension,[19] Patients with ADPKD with DM or MS have earlier age at the diagnosis of hypertension and are associated with poorly controlled blood pressure (Reed). Seeman *et al.* determined a significant correlation between systolic or diastolic blood pressure and the number of and volume of renal cysts.[20]

Helal *et al.* determined that serum uric acid level is associated with the earlier onset of hypertension and increased risk of ESRD independent of BMI and renal functions.[21] Hyperuricemia is an established contributor of decreased blood flow as a result of endothelial dysfunction. Hyperuricemia causes decreased nitric oxide production and increased vascular smooth muscle proliferation, which are associated with endothelial dysfunction.[22] Although hyperuricemia was associated with reduced eGFR, it

Table 6: Distribution of body fat and liquid in male and female participants

			Patients n (%)	Controls n (%)	P
Male	Fat	<20%	17 (50)	12 (50)	0.150
		20–27%	10 (29.4)	11 (45.8)	
		>27%	7 (20.6)	1 (4.2)	
	Liquid	<50%	2 (5.9)	0 (0)	0.476
		50–60%	18 (52.9)	14 (58.3)	
		>60%	14 (41.2)	10 (41.7)	
Female	Fat	<20%	5 (11.4)	0 (0)	0.124
		20–27%	6 (13.4)	4 (12.1)	
		>27%	33 (75)	29 (87.9)	
	Liquid	<50%	25 (56.8)	23 (69.7)	0.228
		50–60%	16 (36.4)	10 (30.3)	
		>60%	3 (6.8)	0 (0)	

Chi-square test; body fat ratio: <20% low, 20–27% normal, >27% high; body liquid ratio: <50% low, 50–60% normal, >60% high.

was not an independent factor for renal progression in ADPKD.[23] Uric acid level is maintained in normal range in individuals with ADPKD with normal renal function. Hyperuricemia and decreased renal excretion of uric acid occur as a result of renal function worsening.[24]

Positive correlation between lipid parameters and BMI or CV mortality was established previously.[25] LDL level is higher than normal in 83% of female and 57% of male individuals with BMI of >25 kg/m^2.[26] A decrease of 1% in LDL is resulted with 2% decrease of coronary heart disease.[27] Abdominal, waist, and hip fat distribution is associated with increased LDL-to-HDL ratio and triglyceride level, which are worse parameters of metabolic control.[28]

Obese individuals have higher incidence of microalbuminuria and proteinuria.[29] Obesity is a well-known risk factor of glomerular disorders. Memili *et al.* found no significant relation between BMI and ESRD.[13] Tirosh *et al.* showed that weight loss improves renal functions[30]. Because approximately 600,000 Americans are affected with ADPKD and more than 2,000 patients are starting dialysis every year, early interventions to slow down the progress are essential in ADPKD[31].

Low sample size and single point measurement of variables were two major limitations of the study. A number of patients with PKD who have exclusion criteria mentioned in Patients and methods section were excluded. Cross-sectional design of the study was another limitation. The importance of the study was; to the best of our knowledge, this was the first report that indicates the association of body fat distribution and insulin resistance in patients with PKD. An interventional study to determine the effect of weight loss on IR in ADPKD may clarify these results.

CVD-related deaths account the most common cause of mortality in ADPKD. There is a strict relationship between IR and CV morbidity.[32] CV events may reversed by early interventions to decrease IR. Physical exercise and life style modifications are recommended to overwhelm CV complications. Further studies with larger sample size will help better understanding of IR in patients with PKD.

Conflict of Interest

The authors declare that there is no conflict of interest.

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